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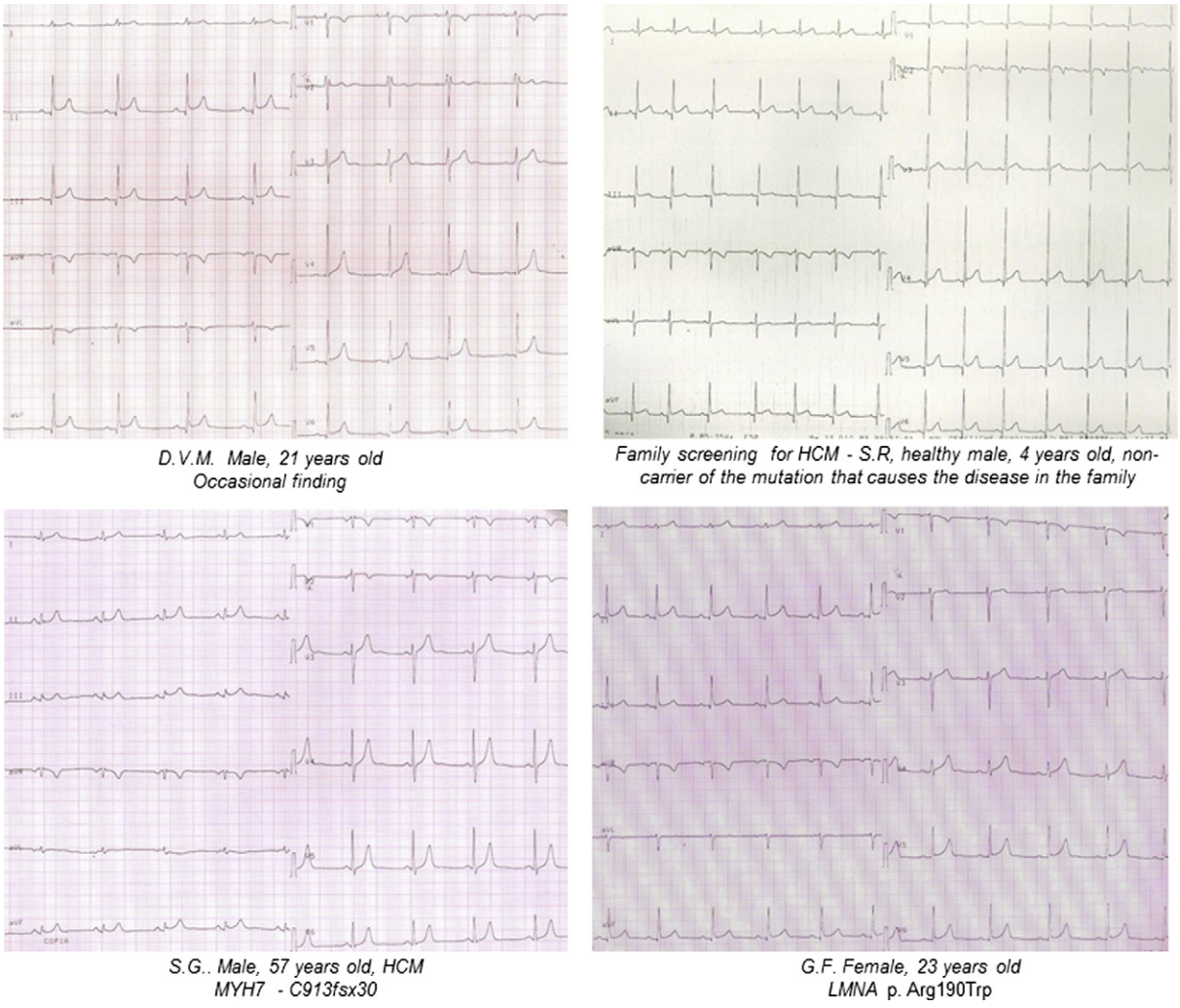

Figure 1 **Different Patterns of J-Point Elevation in Normal individuals and in Patients With HCM and DCM**

DCM = dilated cardiomyopathy; HCM = hypertrophic cardiomyopathy.

Reply

We thank Dr. Serio and colleagues for their interest in our paper (1) and for their letter highlighting the clinical challenge of asymptomatic early repolarization (ER). We noted the examples of J-point elevation in a healthy individual, 2 patients with genetic forms of cardiomyopathy, and a mutation-negative relative. These cases are entirely consistent with our hypothesis that ER may be an independent clinical trait that increases arrhythmic risk in genetically predisposed individuals, but they also illustrate the importance of context when deciding on the clinical significance of ER.

The fundamental clinical distinction is that between physiological J-point elevation, which conveys little or no arrhythmic risk to an individual, and a pathological ER phenomenon that predisposes patients to ventricular fibrillation (VF). In population studies, the presence of a J wave increases the risk of idiopathic VF from 3.4 per 100,000 to 11 per 100,000, making the odds that an individual has a fatal disease 1:10,000, which is too small to influence management (2).

A variety of associated electrocardiogram (ECG) parameters such as the location and amplitude of J-point elevation and a shorter QT interval have been suggested as indicators of an increased risk of VF. However, data from large population cohorts have indicated that the risk is greater in older patients with left ventricular hypertrophy and ischemic heart disease (3,4). These findings need confirmation in larger independent cohorts, and they indicate that other ECG anomalies may be important in determining the sudden death risk of the individual. In addition, further refinement and elucidation of the pattern of ER is still merited.

Based upon our observations and current understanding of the role of ER in VF mechanisms, we suggest that a family history of sudden arrhythmic death syndrome (SADS), epilepsy, or unexplained syncope are new considerations in the interpretation of ER on an ECG because these may indicate either pathological ER or ER in the context of a separate proarrhythmic condition.

In conclusion, at present, ER in the absence of symptoms, structural heart disease, or family history of SADS should not be seen as a major independent risk factor for sudden death. In patients

with a family history of SADS, we would recommend careful evaluation of the pattern and distribution of ER in combination with the ST-segment morphology coupled with investigation for structural or ion channel disease (5). At present, there are no data to support the use of prophylactic antiarrhythmic therapy in cases of isolated ER. However, we would recommend close monitoring and a low threshold for implanting a loop recorder in the context of palpitations, pre-syncope, or syncope. The fundamental questions remain as to what is the prognostic significance, genetic basis, and optimal therapy of ER in the healthy population and high-risk subgroups.

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